on the presence of symptoms (group A: 8 asymptomatic pts and group S: 7 symptomatic pts). We compared pulse pressure, cardiothoracic ratio (CTR), echocardiographic variables (LVDd, LVDs, FS, LAD), plasma ANP and BNP levels between the two groups. The plasma ANP and BNP levels were significantly higher in group S than in group A (107.1 ± 35.6 vs. 21.4 ± 10.8 pg/ml, $p < 0.05$, and 358.0 ± 172.2 vs. 56.5 ± 34.8 pg/ml, $p < 0.05$, respectively). In group A, only one patient showed an abnormal high level of ANP (> 40 pg/ml) while all pts showed abnormal high level of BNP (> 20 pg/ml). On the other hand, all pts in group S showed abnormal high levels of both ANP and BNP. And pulse pressure, CTR, LVDd, and LVDs in group S were significantly increased than those in group A. Pulmonary capillary wedge pressure and left ventricular end-diastolic pressure were also elevated in 4 of 5 symptomatic pts performed cardiac catheterization. These findings suggest that the elevated plasma ANP level indicates the decompensated state of left ventricle and that plasma BNP level is increased not only in symptomatic pts but also in asymptomatic pts with chronic AR according to its severity. *Conclusion:* The measurement of plasma ANP and BNP levels is practical in the evaluation of chronic AR.

1085-41 Plasma Levels of Adrenomedullin (AM) Correlate with the Extent of Pulmonary Hypertension in Patients with Mitral stenosis (MS)

T. Nishikimi, S. Nagata, T. Sasaki, K. Kimura, S. Tomimoto, A. Miyata, K. Kangawa, H. Matsuo, H. Matsuoka, S. Takishita. *National Cardiovascular Center, Osaka, Japan*

AM reduces the blood pressure, pulmonary vascular resistance, and increases pulmonary blood flow. Recent reports showed that AM mRNA and and AM receptor mRNA is strongly expressed in the lung. To determine the pathophysiological significance of AM in the pulmonary circulation, we investigated the relationship between plasma levels of AM and pulmonary hypertension in patients with MS. Plasma levels of AM in blood samples obtained from the vein, pulmonary artery, left atrium, and aorta were measured by RIA in 23 consecutive patients with MS (53 ± 10 yrs) who were undergoing percutaneous mitral commissurotomy. Patients with MS had higher venous plasma levels of AM than age-matched normal controls (3.9 ± 0.3 vs. 2.5 ± 0.3 pmol/L, $p < 0.01$). There was a reduction of plasma AM levels from the pulmonary artery to the left atrium (3.8 ± 0.2 vs. 3.2 ± 0.4, $p < 0.01$). These levels significantly correlated with mean pulmonary artery pressure, total pulmonary vascular resistance, and pulmonary vascular resistance ($r = 0.65$, $r = 0.83$, $r = 0.65$, each $p < 0.01$). Multiple regression analysis revealed that total pulmonary vascular resistance independently correlated with plasma levels of AM ($p < 0.01$). Given that the vascular wall mainly produces AM, and that AM specific receptors exist on the smooth muscle cells, and that AM acts on the pulmonary vascular bed, increased plasma AM in patients with MS may help to prevent the increase in pulmonary arterial resistance in secondary pulmonary hypertension.

1085-42 "Natural Histories" of Mitral Valve Prolapse

A. Zuppiroli, F. Mori, S. Favilli, F. Pieri, V. Troiani, M. Bini, A. Dolara, M.J. Roman [1], M.J. O'Grady [1], R.B. Devereux [1]. *U.O. Cardiologia 2, Ospedale Careggi, Firenze, Italy,* [1] *Cornell Medical College, New York, NY, USA*

The prognosis of mitral valve prolapse (MVP) remains uncertain, as previous longitudinal studies of patients (pts) with MVP give estimates of the incidence of complications from 0.3 to 3.7/100 patient-years (p-yrs). Between January 1979 and August 1996 a total of 263 pts, aged 43 ± 19 years, 140 (53%) women were followed for a mean of 96 months in a referral center for valvular heart diseases. During the follow-up period a total of 63 complications occurred (3.0/100 p-yrs): 46 pts (2.2/100 p-yrs) required mitral surgery, 10 suffered cardiac death (0.5/100 p-yrs), 6 developed necrologic ischemia (0.3/100 p-yrs) and 1 developed infective endocarditis (0.04/100 p-yrs). The overall rate of complications varied significantly (all $p < 0.0001$) in relation to demographic, clinical and echocardiographic variables, being higher in men (odds ratio [OR] = 2.0), in subjects ≥ 45 yrs (OR = 9.5), in those with a holosystolic murmur (OR = 25.4), in those with enlarged (≥ 60 mm) left ventricle (OR = 12.5) or left atrium (≥ 40 mm) (OR = 29.2), in those with a grade 3 or 4 mitral regurgitation (OR = 33.6) and lower in those with an audible midsystolic click (OR = 0.07). This rate of complications far exceeds the 1/100 p-yrs we previously reported in a series of relatively unselected pts or family members in New York, but predictors of complications are quite similar. Therefore it is confirmed that in a common entity like MVP tertiary care centers mainly see the most affected individuals: this selection bias adds to male gender, older age and markers of mitral regurgitation in predicting an adverse "natural history" of MVP.

1085-43 Clinical Substrates for Inducible Monomorphic VT Among Patients with Valvular Heart Disease

C. Narasimhan, Z. Blanck, S. Deshpande, A. Rehman, M.R. Jazayeri, J. Sra, M. Akhtar. *Sinai Samaritan/St. Luke's Medical Centers, Milwaukee, WI, USA*

There is very little data on the electrophysiologic mechanisms and substrates for ventricular tachycardia (VT) among patients with valvular heart disease. We analyzed the mechanism of inducible sustained monomorphic VT in pts with valvular heart disease. There were 27 pts (23 men, 4 women) age 60 ± 15 yrs. In 10 of the 27 pts sustained bundle branch reentry (BBR) was the only mechanism of VT and 17 pts had intramyocardial VT (MVT). Both groups were compared.

	BBR (10)	MVT (17)	p VALUE
1. Age	58 ± 19	60 ± 13	ns
2. Cardiac Arrest	4	5	ns
3. Syncope/presyncope	3	8	ns
4. CAD	2	9	ns
5. EF > 55%	4/10	1/17	< 0.05
6. HV Interval (ms)	85 ± 15	56 ± 14	< 0.01

Conclusion: In pts with valvular heart disease: 1) Sustained BBR is the mechanism of VT in approximately a third of the pts with inducible sustained VT, 2) LV function was better preserved among pts with BBR compared to MVT, and 3) HV interval is significantly prolonged in pts with BBR compared to MVT.

1085-44 Mitral-Valve Prolapse in Military Members: Long-term Follow-up and Clinical Risk Analysis

S.S. Osswald, F.A. Gaffney, J.C. Hardy, W.G. Jackson. *Brooks Air Force Base, San Antonio, TX, USA*

The natural history of mitral valve prolapse (MVP) was assessed in 404 predominantly male, largely asymptomatic military members. Individuals had auscultatory MVP by two cardiologists and/or echocardiographic MVP by 2-D imaging. There were 395 (98%) men and 9 (2%) women. Asymptomatic individuals discovered by routine screening numbered 161 (40%), whereas 243 (60%) were cardiovascular (CV) referrals. The ages ranged from 21 to 64 years (mean = 36 years). Follow-up time averaged 8.6 years (1–21 years). During follow-up, 5 patients died, 3 from non-cardiac causes, 5 required mitral valve surgery, 4 experienced cerebral ischemic events, 7 progressed to severe MR, 10 had episodes of atrial fibrillation, and 8 had SVT greater than 15 seconds. There were no episodes of endocarditis or ventricular tachycardia (> 15 sec). SBE prophylaxis was reported in 77% of individuals by survey. The overall rate of significant complications was 0.92%/year. Stepwise logistic regression selected left ventricular enlargement (LVE) > 6 cm (adjusted odds ratio (OR) = 7.7, $p < 0.001$), left atrial enlargement (LAE) > 4 cm (adjusted OR = 4.3, $p = 0.004$), and age > 45 years (adjusted OR = 3.2, $p = 0.015$) to be significant predictors of complications. In univariate analysis, additional risk factors of mitral valve thickening with prolapse and CV referrals both had odds ratios significant to $p < 0.05$. *Conclusion:* The risk of complications with MVP in an asymptomatic, male population is low. Significant risk factors include LVE, LAE, and age > 45 years. Risks also appear higher for those referred for CV diagnosis and those with both mitral valve thickening and MVP on echocardiogram. Auscultatory MVP was not a significant risk factor.

1086 Detection of Cardiac Transplant Rejection

Wednesday, March 19, 1997, 3:00 p.m.–5:00 p.m.
Anaheim Convention Center, Hall E
Presentation Hour: 3:00 p.m.–4:00 p.m.